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Sidra Sattar

Liaquat national hospital Karachi

Saba Zaidi

Liaquat national hospital Karachi

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A CASE OF HEROIN INDUCED TOXIC LEUKOENCEPHALOPATHY

Sidra Sattar¹, Saba Zaidi²

¹Resident emergency medicine

²Assistant Professor Neurology at LNH

Institute: Liaquat National Hospital

Correspondence to: Sidra Sattar, Resident emergency medicine Email: sidra.sattar91@gmail.com

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ABSTRACT:

Heroin-induced leukoencephalopathy comes under the category of Toxic leukoencephalopathies. We report the case of middle-aged gentleman with the history of polysubstance abuse who presented with recurrent falls, difficulty in walking and drowsiness. Diffuse white matter changes were appreciated on MRI Brain with sparing of cerebellum. In context of his history of inhalational heroin abuse with extensive symmetrical white matter changes in cerebrum, a diagnosis of heroin induced toxic leukoencephalopathy was made.

INTRODUCTION:

Leukoencephalopathy is a syndrome of neurologic deficits including alteration of mental status caused by pathological changes in cerebral white matter. The term "Toxic leukoencephalopathy" include a wide range of toxins and clinical presentations. Toxic leukoencephalopathy secondary to heroin was first described by Wolters in 1982 after an outbreak in Netherlands⁽¹⁾. Since then number of cases have been reported. Diagnosis requires neurobehavioral deficits, exposure to toxin and specific MRI Brain changes suggestive of white matter disease. Our case fulfilled the diagnostic criteria for toxic leukoencephalopathy. Other causes of leukoencephalopathy have been excluded by detailed history and thorough investigations.

BACKGROUND:

Among the opioids Heroin is the most common illicit drug of abuse and has significant neurological adverse effects.⁽²⁾ Mechanism of action includes activation of three types of receptors mu, kappa and epsilon. Stimulation of mu receptor results in respiratory depression, miosis, euphoria and respiratory depression while stimulation of later two contributes to psychomimetic effects and dysphoria⁽³⁾

Heroin can be abused either in injection, oral or inhalational form. "Chasing the dragon" refers to inhaling the smoke(pyrolysate vapors) from heated heroin on a piece of foil⁽⁴⁾ There is some variability in the range of neurological complications related to route

of intake. Literature review revealed that intravenous heroin abuse is associated with hypoxic-ischemic encephalopathy while inhalational heroin is more related to toxic leukoencephalopathy pathologically termed as spongiform leukoencephalopathy.^(5,6)

Clinical presentation varies from stupor, coma and death after a latent period of hours days to months following exposure of heroin. Three stages frequently described in literature. Clinical features of initial stage include motor restlessness, pseudobulbar speech and cerebellar ataxia followed by intermediate stage which includes rapid worsening of cerebellar symptoms, hyperactive reflexes, spastic hemiplegia or quadriplegia, myoclonus and chorea.⁽⁷⁾

Pathologically there is spongiform degeneration of white matter especially the corticospinal and solitary tracts with associated multivacuolar degeneration of oligodendrocytes on histopathology.^(8,9)

Neuroimaging is pathognomic which include cerebellar white matter, cerebellar and cerebral peduncles, posterior limb of internal capsule and centrum semiovale with sparing of U-fibres.^(10,11) Symmetry of signals points towards toxic or metabolic etiology. Radiological sparing of infratentorial region is seen in our patient. The exact cause is unknown. Blasel et al described cases in which there was only supratentorial involvement and sparing of infratentorial structures. Similarly a recent case report from India showed sparing of infratentorial structures as well. In all these

above mentioned cases the heroin was abused in inhalational form.^(10,11)

CASE REPORT

34 year old gentleman with the ten years history of polysubstance abuse predominantly inhalational heroin, alcohol and benzodiazepines presented with history of gait imbalance, frequent falls, dysarthria and drowsiness for 2 weeks. Brothers narrated this history and added that the patient used to stay outside home for the most of his time. He is married with four children. He never took any responsibility as far as his family or home is concern. His Children were taken care of by their paternal uncles. In between these ten years he tried to leave addiction once or twice. He was habitual of taking hand full of benzodiazepines. These drugs added aggression to his personality to an extent of physical and verbal abuse. On examination in clinic he was drowsy but arousable on verbal command. Had severe dysarthria. Moving all four limbs spontaneously. Reflexes were brisk. No clonus. Plantars were flexor. He was kept on benzodiazepines and antipsychotics for control of withdrawal symptoms.

MRI brain with contrast showed diffuse symmetrical signals supratentorially involving white matter sparing cerebellum and brainstem. Predominantly on T2 and Flair images. No diffusion restriction. No post contrast enhancement.

CSF studies were normal. His antipsychotic medications were adjusted and antibiotic was started for possible aspiration pneumonia. HIV and VDRL found to be negative. Neurological assessment following above management showed significant improvement in mentation. At that time neurology team tried to do certain cognitive tests. Overall impression was labile mood, inappropriate replies, failed to do serial 7 and positive primitive reflexes. Cranial nerves were intact grossly. Action fine tremors were appreciated in all four limbs. Tone was increased. Reflexes remained exaggerated. Gait was apraxic and broad based. Patient's attendants were guided regarding changes in MRI brain and variable prognosis. Strict abstinence from Heroin was advised. He was discharged home with rehabilitation measures.

DISCUSSION:

The group of drugs collectively includes heroin, morphine, hydrocodones, oxycodone, hydromorphone, codeine as well as other narcotics such as phentanyl, meperidine and opium.¹³ When this particular group is used for medical purposes, the most important role is as pain killer, which comes from activation of kappa

and delta receptors with combined effects including euphoria and drowsiness, this is typically from activation of opioid Mu receptors located at the nucleus accumbens, this particular characteristic of the group is famous among drug abusers who seek euphoric effects while attempting to avoid withdrawal sideeffects.^{14,15}

Heroin, a diacetyl derivative of morphine, with its common usage among drug addicts varies in its form of consumption for instance; intravenously, subcutaneously, smoked, sniffed or inhaled. When talking about common medical related complications in addition to is psychosocial degeneration are encephalopathy, transverse myelitis, myelopathy, cerebral infarctions, meningitis, cerebral abscess, mycotic aneurysms, myopathies and plexopathies.¹⁶

The clinical spectrum has been defined to vary over a wide range from a more favourable outcome to culmination to death in approximately 25% of reported cases.¹⁷ In the acute stage neurologic symptoms are more prevalent for example motor restlessness, apathy, bradyphrenia, cerebellar speech disturbances and ataxia, this may be followed by an intermediate stage characterized with rapid worsening of cerebellar symptoms with prominence of exaggeration of deep tendon reflexes and additional pathologic reflexes, spastic hemiparesis, tremor and myoclonic jerks or choreoathetoid movement disorders.¹⁸ Finally, the terminal stage may consist of vegetative dysregulation spasmodic stretching, hypotonia and akinetic mutism.¹⁹ This particular pattern of symptomatology has been observed in our case as well.

A rare complication of inhalational form of heroin can be spongiform leucoencephalopathy, reported first by Wolters et al.¹⁷ in 1982, though this complication is very scarce in literature only 56 cases been reported in literature and has mainly been related to the additives that becomes active after being burnt and pertains to its toxic effects, this was seen in our patient using inhalational form of abuse of this drug. These additives as defined in literature are, "caffeine, phenobarbitone, methaqualone, procaine, piracetam, and lignocaine".²⁰

This phenomenon of leucoencephalopathy has been so far mainly been associated to inhalational form of abuse. A reason explaining such phenomenon could be related to the dose of intake, as it is more when using the inhalational form by abuser, whereas other forms of abuse may not be the culprit so far.²¹

The histopathological distinction has been defined as "vacuolar degeneration of cerebral white matter tracts", when viewing with electron microscopy it appears as "fluid entrapment between myelin lamellae and

absence of demyelination in these patients."²²

Magnetic resonance imaging(MRI) when performed in such patients shows bilateral symmetrical hyperintense lesions in the white matter of cerebrum and cerebellum on T2-weighted images. Blasel et al, defined mainly cerebral involvement and complete sparing of infratentorial structures in his three patients, this pattern was observed in our patient too.²³

CONCLUSION:

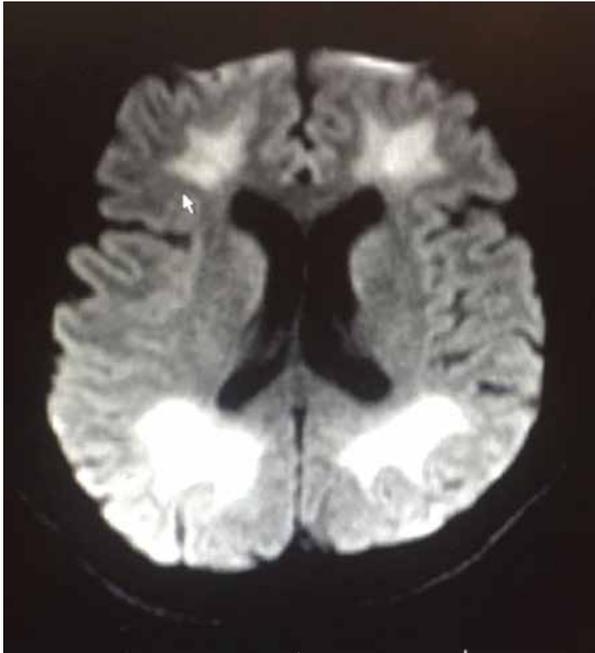
We presented a rare case of heroin induced toxic leukoencephalopathy.

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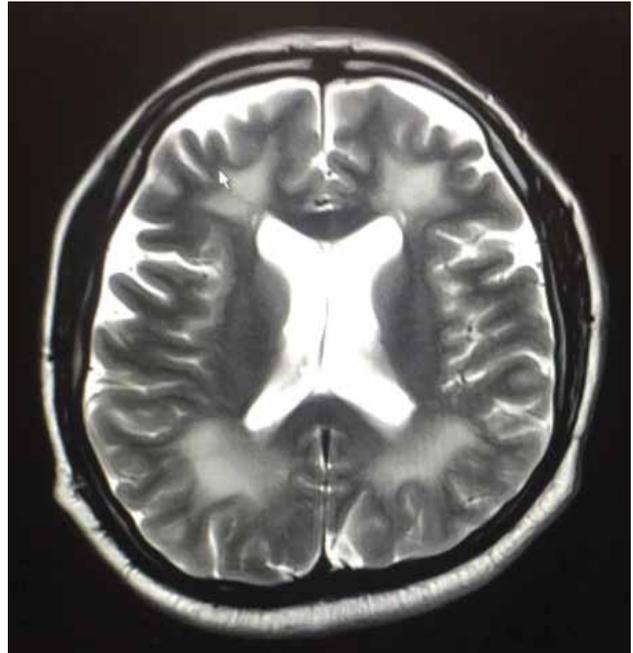
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MRI BRAIN FLAIR SEQUENCE SHOWING PERIVENTRICULAR HYPERINTENSITIES



MRI BRAIN T2 SEQUENCE (SYMMETRICAL PERIVENTRICULAR HYPERINTENSITIES)

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Sidra Sattar; concept, data collection, data analysis, manuscript writing, manuscript review

Saba Zaidi; data collection, data analysis, manuscript writing, manuscript review